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Advances in the Study and Treatment of Acute Myocardial Infarction

THE CLINICAL IMPORTANCE of acute myocardial infarction in producing disability and death is emphasized dramatically by the estimates which suggest that in the United States alone, in 1971, more than 1,000,000 persons will have myocardial infarction and more than 250,000 of them will die of the attack. Moreover, one-third of these patients will be under the age of 65 years. These facts suggest the need for greater research on this important disease. While most research, at present, is directed toward better diagnostic and therapeutic approaches in patients with coronary artery disease, there is little indication that these studies will affect mortality significantly. Innovative studies into the causes of acute infarction and to the predisposing causes of atherosclerosis are needed.

In the Speciality Conference, "Research on the Diagnosis and Treatment of Myocardial Infarction," by Braunwald and associates published in this issue of CALIFORNIA MEDICINE, much attention is given to new clinical and laboratory features of acute myocardial infarction.[1] Emphasis is directed toward early diagnosis and the possibility of surgical intervention. Additional publications[2,3] have recently discussed the possibility of reducing the morbidity and mortality from acute myocardial infarction by surgical techniques. Hence this review of the newer diagnostic and prognostic advances seems timely.

In establishing the diagnosis of acute myocardial infarction, the electrocardiogram remains one of the primary methods available to physicians, although confirmation by measuring alterations in serum enzymes and for the detection of smaller areas of necrosis not usually producing diagnostic electrocardiographic changes are important. In the specialty conference, Dr. Karliner describes two new techniques for detecting myocardial necrosis. Detection of elevations of glyceraldehyde phosphate dehydrogenase (GAPDH) in patients with acute myocardial infarction appears to be highly reliable and technically simple. The great advantage over detection of elevations in SGOT and LDH is that GAPDH is elevated earlier and remains elevated for only a short time. This allows the detection of a recurrent infarction, or extension of the initial area of necrosis. Measurement of isozymes of LDH, which is also discussed, potentially has advantages but to date no convincing evidence is available that these enzyme fractionations will lead to improved diagnosis.

Various methods to determine what areas of the myocardium are inadequately perfused have been devised recently. Coronary arteriography is the most direct method, but because of the risk to patients with acute infarction, it is not frequently employed. The techniques utilizing injections of microaggregated radioactive labelled albumin into the coronary arteries for external scanning with a detector is of interest. The technique needs further quantitation, however, and does not remove the need for catheterization of coronary arteries. Its risks, when widely employed, are not known and it fails to delineate precisely the anatomical changes in the coronary arteries, so that surgical intervention can be considered.

Radarkymography allows the physician to detect areas of ventricular wall which contract in an abnormal manner (aneurysms) in acute and chronic myocardial infarction. This technique is another of the non-invasive methods available for studying ventricular performance, and its clinical value in patient study may well be great after appropriate validation.

In order to evaluate better the late clinical course of patients following acute myocardial infarction, quantitation of the size of the infarction is needed. Determination of ventricular size as described by Ross in this published specialty

clinic allows determinations of left ventricular size which is correlated with a higher incidence of late deaths. Only more clinical experience with this technique will clarify its place in the care of patients with acute myocardial infarction.

A new and interesting technique to determine the size of an infarcted area of myocardium has been developed by Dr. Maroko and colleagues. It is based on determining the area of injury by utilizing direct electrode recordings from the myocardium and quantitating the degree of ST segment elevation.[1] This technique has been studied in animals with experimental coronary artery occlusion and has been correlated with the decline of creatine phosphokinase in the ischemic tissue. Utilizing these techniques to quantify the degree of myocardial damage, the effects of glucagon, beta-blocking agents, digitalis glycosides and anti-arrhythmic agents on infarcted tissue have been studied.

While all of these techniques are of interest in the laboratory, they cannot be applied in man. In addition, a number of questions regarding the value of studying ST segment elevation for quantifying myocardial damage have been raised.[4] These include the detection of subendocardial and intramural myocardial infarction, the need to apply pressure to the myocardium by electrodes which alter ST elevations, and the difficulty in placing electrodes precisely for repeated study.

The early surgical intervention in patients with acute myocardial infarction who develop rupture of papillary muscles, rupture of the ventricular septum and large ventricular aneurysms has become commonplace.[5,6] The results of such operations have been good and they depend to a large extent on the severity of myocardial damage in each individual patient. The resection of infarcted tissue and the use of saphenous by-pass grafts from aorta to coronary artery are being carried out under experimental protocols in patients with acute myocardial infarction. These techniques need further evaluation before being adopted widely.

Although much of the investigation described by Braunwald and colleagues is promising, many more studies with these and other new methods are necessary before clinical care of patients with acute myocardial infarction can be expected to improve. In addition, the development of new methods for studying and treating patients after symptomatic disease develops will likely have only a small effect on mortality from this disease process.

Among the several fields of investigation which have promise for reducing mortality are the identification and treatment of high-risk individuals, understanding of genetic abnormalities of lipid metabolism, public education regarding risk factors and the desirability of seeking early medical attention after infarction, and the development of effective circulatory assist devices. One other area of particular promise is the identification of precipitating factors in acute infarction.[7] While acute infarction and sudden death have both increased drastically, there is less substantial evidence that atherosclerosis has increased dramatically.[7,8] The factors which precipitate acute infarction may be identified by studying intently patients with impending infarction. These studies must include assessment of metabolic, hematologic, social and psychological, environmental and cardiac factors related to infarction. While no dramatic breakthroughs appear on the horizon, substantial long term progress appears possible.

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Of Pharmacists, Physicians And Health Care

In his wide ranging discussion which appears elsewhere in this issue Dean Goyan forthrightly addresses himself to many problems in patient care which he recognizes as going far beyond